PATENT

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application No.:	10/535,215
Confirmation No.:	2007
Filing Date:	May 17, 2005
Applicants:	Jan SVENSSON et al.
Title:	FUNCTIONAL CEILING SYSTEM
Attorney Docket:	315-0039/US

Customer Service Window
Randolph Building
401 Dulany Street
Alexandria, VA 22314
Mail Stop PETITION

September 11, 2009

PETITION TO REVIVE UNDER 37 CFR § 1.137(b)

Sir:

Applicants petition for revival of the above-identified application on the ground that the application was unintentionally abandoned by failing to respond to the January 12, 2006 Missing Requirements Notice by August 12, 2006. Based upon information and belief, the undersigned attorney states that the abandonment was unintentional, and also states that the entire delay in filing the required reply from the due date for the reply until the filing of this Petition was unintentional.

To show that the entire delay was unintentional, the following two Statements are concurrently submitted in connection with this Petition.

1. A Statement by Mr. Steven S. Payne (including EXHIBITS A-C), which discusses the cause of the delay in reply that originally resulted in abandonment up to September 4, 2009; and
2. A Statement by Mr. Herman R. Heflin III, which discusses the cause of the delay from September 4, 2009, until the filing of this Petition.

Applicants concurrently submit the reply to the January 12, 2006 Missing Requirements Notice.

Applicants also concurrently submit the Petition fee of $1,620.00 under 37 CFR § 1.17(m). Please charge any additional fees or credit any overpayment to Deposit Account No. 50-4446.

Respectfully submitted,

/Herman R. Heflin III/
Herman R. Heflin III, Reg. No. 41,060

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STATEMENT BY MR. STEVEN S. PAYNE

Sir:

In support of the Petition to Revive Under 37 CFR § 1.137(b) submitted concurrently herewith, please consider the following information.

1. I, Steven S. Payne, am the attorney who represented Applicants in filing United States Patent Application No. 10/535,215 ("the '215 application") on May 17, 2005.

2. A Notification of Missing Requirements was mailed January 12, 2006.

3. I did not file any response to the January 12, 2006 Notification of Missing Requirements, and a Notice of Abandonment was mailed on September 5, 2006.

4. I, as a sole practitioner, had the sole right to reply to the January 12, 2006 Notification of Missing Requirements, and the right to revive the '215 application after the abandonment.

5. The reasons for my failure to file a response to the January 12, 2006 Notification of Missing Requirements and for not filing a petition to revive the application in a timely fashion, I believe can be attributed to my mental state during the time in question.

6. In 2003, I was diagnosed by Dr. S. Mark Tanen with a Thyroid disease, specifically Hashimoto's disease. In Hashimoto's, antibodies react against proteins in the Thyroid gland causing gradual destruction of the gland itself and making the gland unable to

produce the thyroid hormones the body needs. As a result, I have been taking Synthroid, a thyroid replacement hormone, everyday. Since 2003, my thyroid condition has continued to gradually deteriorate and Dr. Tanen has had to raise my daily dosage of Synthroid several times.

7. It is well known that some of the most profound effects on thyroid hormone imbalance are in the mental arena. Some people with Hashimoto's disease may sleep easily but do not get full refreshment from their sleep. During waking hours, they experience fatigue, apathy and "brain fog" (short-term memory problems and attention deficits). These problems may affect their daily functioning and cause increased stress and depression. See EXHIBITS A-C for more background on thyroid disorders.

8. The usual treatment for Hashimoto's is taking thyroid hormones in pill form such as the Synthroid I was prescribed.

9. I slowly but steadily started to experience the mental issues such as apathy, brain fog and depression. But since they did not occur quickly but rather developed slowly over time, I did not recognize nor associate these problems with my thyroid disorder. I am also a very private person so I did not share these problems with my family, Dr. Tanen or anyone. I simply maintained a facade that all was well even though some days at work would simply fly by in a fog and nothing would be accomplished.

10. Through 2005-2007, my condition continued to worsen and my depression grew profound. Many work related things were late or missed because of my lack of concentration and the depression I was suffering. Since I am a sole practitioner, there were no colleagues to notice the problems.

11. In January of 2008 and again in April of 2008, I attempted suicide.

12. After the second suicide attempt, I could no longer maintain my façade and my family demanded to know what was happening with me. As a result, I told my family and my clients what had been going on.

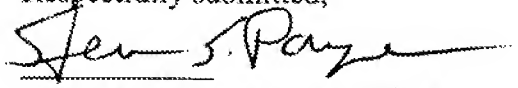
13. I also went to Dr. Tanen to tell him what was happening. After hearing of my condition, he told me that he wished I had come to him and told him about these symptoms when they first started to occur. There are two types of thyroid hormones: L3 and L4. Synthroid is an

L4 hormone replacement. While the use of Synthroid is the usual treatment for Hashimoto's disease, some people who are experiencing the same mental problems I was experiencing while just taking Synthroid have found that a combination of L3 and L4 replacement hormones can greatly reduce the mental problems associated with this disease. Doctors are reluctant however to prescribe L3 to patients that are not experiencing the mental problems associated with the disease because L3 hormones can cause damage to the heart. With Dr. Tanen's help, I am now on a combination of prescription drugs Synthroid (L4) and Cytomel (L3) and I am enjoying a reduction of my symptoms. For brevity I have not gone into the complicated medical theory regarding thyroid disease and the differences between L3 and L4 replacement hormones but have attached EXHIBITS A-C in support for my statements above. I would particularly recommend EXHIBIT A, the article entitled "Use of T3 Thyroid Hormone To Treat Depression" by Dr. Gabe Mirkin.

14. My failure to respond to the January 12, 2006 Notification of Missing Requirements, and promptly file a petition to revive the application was unintentional and was due to the mental state I was experiencing during the time in question.

15. I declare that all statements made herein of my own knowledge are true, and that all statements made on information and belief are believed to be true. These statements were made with knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Respectfully submitted,



Steven S. Payne, Reg. No. 35,316

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EXHIBIT A

USE OF T3 THYROID HORMONE TO TREAT DEPRESSION

Gabe Mirkin, M.D.

If you are tired much of the time, your doctor will order blood tests for the two thyroid hormones called T3 and T4 and for the brain hormones called TSH and prolactin. If your TSH is high and your prolactin is normal, you are probably hypothyroid and need to take thyroid hormone to give you more energy and prevent heart and blood vessel damage.

Doctors treat people with low thyroid function with thyroid pills called T4 (Levothroid, one brand name is Synthroid). Many doctors think that a person needs only T4 because the thyroid gland makes T4 and then it is converted to T3 in other tissues. However, some people become depressed when they take just T4 and their depression can be cured when they take both thyroid hormones, T3 and T4.

When a depressed patient comes to me and is taking thyroid hormone, T4, I immediately order a blood test called TSH to check if he or she is getting the correct dose. If the TSH is normal, I reduce the dose of T4 by 50% and add a very low dose of T3 (brand name, Cytomel) because it safer to prescribe too low a dose, rather than too high a dose. Overdoses cause shakiness, irritability, irregular heart beats, diets, and osteoporosis. The patient returns in one month for a blood test, TSH, to see if the total thyroid dose is correct. If the TSH is too high, the thyroid dose is too low and I raise the T3 (Cytomel) dose by 5 to 10 mg each month until the TSH is normal. Then once a year I check TSH blood levels to make sure that the person's requirements for thyroid hormone are being met.

For example, the usual replacement dose for low thyroid function is 100 micrograms per day. If a depressed patient has a normal TSH, I reduce the T4 dose to 50 mcg/day and add 5 mcg of T3 per day. One month later, if the TSH blood is still too high I raise the T3 dose to 10 or 20 mcg and continue to increase the T3 level each month until the TSH is normal.

Exciting research shows that the thyroid hormone called T3 can help treat depression (1,2,3). Psychotherapy often fails to control depression. Sigmund Freud, the father of psychotherapy, proposed theories about depression, that many psychiatrists do not accept because his writings were his opinions and not presented as scientific data supported by controlled experiments. The dominant theory today is that depression is caused by low brain levels of the neurotransmitters, serotonin and norepinephrine. The drugs such as Paxil, Prozac and Zoloft that treat depression are supposed to raise brain levels of these neurotransmitters. Doctors can also raise brain levels of serotonin by prescribing pills containing T3, a hormone produced by peripheral tissue from T4, which is produced by the thyroid gland. (1) They also prescribe T3 by itself or together with antidepressants. Depression is common among people who have too much or too little thyroid hormone. Doctors usually treat low thyroid function with T4 also known as Levothroid and many people become even more depressed. They treat this depression by prescribing T3 as well as T4.

An article in the Journal of Clinical Psychiatry shows that T3 can be used to treat post traumatic stress disorder, commonly seen in soldiers and people who have been through other causes of terrible emotional trauma (13).

Try to balance T3 and T4 so you will not be taking too much thyroid and harm yourself. 1)If you now take 100 mcg of Levothroid (T4): 2) Lower T4 (Levothroid) to 50 mcg and add Cytomel (T3) 5 mcg each day. 3) One month later, have your doctor draw blood for TSH. 4) If it is normal, you are on the correct dose and should get blood tests TSH once a year. 5) If TSH is too high, increase Cytomel to 10 mcg and hold Levothroid at 50. 6) Draw monthly TSH until it is normal. Keep on raising Cytomel by 5 mcg until TSH is normal.

1) M Weissel.Treatment of psychiatric diseases with thyroid hormones.Acta Medica Austriaca, 1998, Vol 26, Iss 4, pp 129-131.

2) H Meyer, MKH Schafer, K Bauer.Thyrotropin-Releasing Hormone (TRH), a signal peptide of the central nervous system.Acta Medica Austriaca, 1998, Vol 26, Iss 4, pp 119-122.

3) F Konig, C von Hippel, T Petersdorff, W Raschka.Antithyroid antibodies in depressive diseases.Acta Medica Austriaca, 1999, Vol 26, Iss 4, pp 126-128.

4) A Steiger.Thyroid gland and sleep.Acta Medica Austriaca, 1999, Vol 26, Iss 4, pp 132-133.

5) Jackson IM .Thyroid 1998 Oct;8(10):951-6.

6) Refractory depression: treatment strategies, with particular reference to the thyroid axis. Joffe RT . J Psychiatry Neurosci 1997 Nov;22(5):327-31 .

7) Thyroid hormones in depressive disorders: a reappraisal of clinical utility. Lasser RA , Baldessarini RJ. Consolidated Department of Psychiatry, Harvard Medical School, Boston, Mass., USA. Harv Rev Psychiatry 1997 Mar-Apr;4

(6):291-305

8) The hypothalamic-pituitary-thyroid axis in major depression.Sullivan PF , Wilson DA , Mulder RT , Joyce PR University Department of Psychological Medicine, Christchurch School of Medicine, New Zealand. Acta Psychiatr Scand 1997 May;95(5):379-8

9) S Ramschak Schwarzer, W Radkohl, C Stiegler, MP Osmai, G Leb. Interaction between psychotropic drugs and thyroid hormone metabolism - an overview. Acta Medica Austriaca. 2000, Vol 27, Iss 1, pp 8-10.

10) Dorn et al. Baseline thyroid hormones in depressed and non-depressed pre- and early-puberty boys and girls. J Psychiatry Research 1997(Sept_Oct);31(5):555-67.

11) Birkenhager TK et al. An open study of triiodothyronine augmentation of tricyclic antidepressant in inpatients with refractory depression. Pharmacopsychiatry 1997(Jan);30(1):23-26.

12) SR Rack, EH Makela. Hypothyroidism and depression: A therapeutic challenge. Annals of Pharmacotherapy, 2000, Vol 34, Iss 10, pp 1142-1145.

13)Triiodothyronine augmentation of selective serotonin reuptake inhibitors in posttraumatic stress disorder. O Agid, AY Shalev, B Lerer. Journal of Clinical Psychiatry, 2001, Vol 62, Iss 3, pp 169-173.

Checked 3/8/09

EXHIBIT B

Thyroid Hormone Disorders

(Released May 2001)

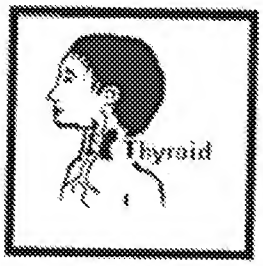
by Jennifer A. Phillips

Review Article

Introduction

Hormones are named from the Greek word *hormon*, meaning "to urge or excite", because they were first discovered to play a role in hunger, sex, flight-or-fight response, and many other basic drives. Hormones serve within the body as invaluable messengers, governors of development, and regulators of metabolism. This Hot Topic will focus on the effects of thyroid hormone (TH) and the disorders that are associated with TH imbalance.

TH, found in all chordate animals, is the only major biochemical molecule known to incorporate iodine, a substance common in the sea but rare on land. Iodine is essential to the structure of TH, and iodine deficiency is the leading cause of hypothyroidism in undeveloped countries. TH is produced by the thyroid, a butterfly-shaped gland behind the larynx, in response to thyroid stimulating hormone (TSH), which is released by the pituitary gland.



TH exists in two major forms. Levothyroxine (T4), with four iodine atoms per molecule, is an inactive form that can be converted into T3, and is produced exclusively by the thyroid gland. Triiodothyronine (T3), with three iodine atoms per molecule, is eight times more effective than T4. It is converted from T4 in the thyroid, brain, liver, and bloodstream, and in various tissues of the body.

The Role of TH in the Body

One important function of TH is helping the body convert food into energy and heat. T3 directly boosts energy metabolism in mitochondria, the powerhouses of cells. T3 triggers rapid protein synthesis and influences mitochondrial gene transcription, the reading of genes and synthesis of proteins from genetic information. These activities cause breakdown of proteins and an increase in free fatty acids, as well as increased oxygen use. TH elevates the heart rate to meet the increased oxygen needs.

TH also regulates body temperature. TSH, which stimulates the thyroid to produce TH, also stimulates brown adipose tissue, a mitochondria-rich tissue, to boost heat production in mammals without muscle activity. TH fluctuates in response to caloric intake and external temperature. During starvation, the body naturally lowers TH, not only to reduce caloric needs, but also to prevent ketone bodies from building up in the blood and kidneys. Ketone build-up, which can also happen in diabetes, can cause damage to the kidneys and other part of the body. Injury and illness lower TH levels, which rebound once the patient is healed.

TH is sensitive to the levels of other hormones besides TSH. Estrogen partially blocks the efficiency of TH, so women compensate by producing more TH than men. This may be why women have larger thyroids than men and are more prone to thyroid disease of all types. Women who take TH replacement pills must increase their TH dosage if they start taking birth control pills, to compensate for the higher levels of estrogen from birth control pills. Growth hormone also partially blocks TH, but it also complements TH in its effects on growth,

development, and metabolism.

TH plays a major role in metamorphosis and development in all vertebrates. It affects development by binding to thyroid hormone receptors (TRs), molecules that then change their shape to an activated form. Once activated by TH, TRs can bind to responsive elements in the DNA, triggering gene transcription. The position of the TR attaching to the responsive elements facilitates the copying of some genes, and blocks others from being copied. Two major forms of thyroid hormone receptors exist: TRα and TRβ.

TRs are nuclear receptors like retinoid A receptors, Vitamin D receptors, and steroid hormone receptors. TRs change configuration when attached to T3, and this changed configuration allows them to attach to responsive elements in the genome. Nuclear receptors are often dimerized (attached to another nuclear receptor of the same or different type), but they remain inactive until bonded by the usual trigger. For example, thyroid hormone receptors dimerized with retinoid X receptors will not activate until they are bonded with T3 or retinoids (derivatives of Vitamin A).

We still do not know all the genes that are regulated by TH. Some TR-responsive elements in the DNA are Alu elements, which are able to move around in the genome on occasion, creating even more Alu elements in the genome. This allows many different genes to come under the control of TH without the genes themselves mutating. Different species may have different genes under control of TH, especially those concerned with development. For instance, while most mammals show similar symptoms of hypothyroidism (fatigue, apathy, etc.), dogs show the additional symptom of seizures. Most chemicals that cause hypothyroidism do not block thyroid receptors in the genes; they only block the efficiency or synthesis of TH. Hence most of our information about which genes are regulated by TH comes from studying genetic disorders in which the TRs are non-functional.

Genetic Disorders Involving TH, TSH, or TRs

Resistance to TH is a genetic disorder caused by mutations in the TRb gene. Patients with this disorder have high TH levels and TSH levels, goiter (enlarged thyroid gland), and mild hypothyroid metabolisms. Clinical effects are less severe than with congenital hypothyroidism and can include short stature, delayed bone maturation, hyperactivity, learning disabilities, and hearing defects, as well as mixed features of hyper- and hypothyroidism. This condition is usually inherited dominantly.

Pendred's Syndrome is caused by a genetic defect that limits the incorporation of iodine into thyroid hormone, which wrecks the structure of the hormone. Pendred's Syndrome can cause hypothyroidism with goiter. The body compensates by producing more TSH and working harder to make enough thyroid hormone that works. The syndrome can also cause more serious problems, such as profound deafness, or non-syndromal deafness alone. These symptoms are present from birth. People who develop hypothyroidism later in life may have ringing in their ears and dulled hearing, but these symptoms are usually correctable by TH therapy, while deafness caused by Pendred's Syndrome is not.

TSH receptor (thyrotropin receptor) gene mutations often cause hyperthyroidism, or TSH insensitivity, which leads to normal TH levels in the blood with elevated TSH levels. TSH has unknown effects on lymphocytes and brain cells; therefore imbalances affecting TSH levels may cause additional, unknown effects on the brain and immune system. One mutation was found in association with Graves' disease. Graves' disease is an autoimmune form of hyperthyroidism, and the genes that seem to increase risk of Graves' disease are associated with immunity.

In humans, thyroid hormone plays a notable role in brain development from the middle of pregnancy to the second year of life. Maternal or fetal hypothyroidism, whether caused by lack of iodine during the pregnancy, or by other problems, can cause a non-genetic condition called cretinism. Babies affected by cretinism can develop normal intelligence if the condition is remedied within a few months, but otherwise they suffer severe, irreversible mental retardation. One severe type of cretinism can also be caused by mutations in the TRα gene, and may be untreatable.

Effects of TH Imbalance: Hypothyroidism

Some of the most profound effects of TH imbalance are in the mental arena. Hypothyroid people sleep easily and do not get full refreshment from their sleep. During waking hours, they

experience fatigue, apathy, and "brain fog" (short-term memory problems and attention deficits). These problems may affect their daily functioning and cause increased stress and depression.

TH acts as a neurotransmitter. TH imbalance can mimic psychatric disease because T3 influences levels of serotonin, a neurotransmitter integral to moods and behavior. Low levels of T3 can cause depression. Some anti-depressants make hypothyroid patients feel even worse because the medications depress T3 levels. Paradoxically, some substances labelled depressants such as alcohol or opiates can increase T3 levels by impairing the breakdown of T3 in the brain, thus lifting mood. This may be one reason why these substances are so addictive.

Severe hypothyroidism can cause symptoms similar to Alzheimer's disease: memory loss, confusion, slowness, paranoid depression, and in extreme stages, hallucinations. Thyroid disease is one of the many treatable diseases that must be ruled out before arriving at the diagnosis of Alzheimer's, which is incurable and cannot be definitely diagnosed until after death. Risk of hypothyroidism increases with age; by age 60, 17% of women and 9% of men have symptoms of thyroid disease[5].

Low TH levels also produce fatigue, slight hypoglycemia (low blood sugar), slowed digestion of food, and constipation. Infertility is common. These symptoms can indicate that other diseases are present, particularly because TH levels tend to go down during prolonged illness in an effort to conserve energy. Chronic disease, such as Lyme disease, can mimic (or cause) hypothyroidism. Hypothyroidism is not difficult to diagnose by symptoms, if the patient reports enough symptoms to the doctor and if the doctor thinks of it. Diagnosis can be confirmed by blood tests, but the cause is less easy to discern.

TH imbalance has a profound effect on cardiovascular fitness because TH helps control heart rate and blood pressure. Under hypothyroid conditions, the heart can slow to 30 heart beats a minute and develop arrhythmia. Blood pressure may fall from normal levels of 120/90 to 70/50. Hypothyroidism also weakens muscles, including the diaphragm. As a result, breathing can become less efficient. A goiter impairs breathing even more. Snoring may start or become worse. Fatigue sets in easily; in fact it never quite leaves a person with symptomatic hypothyroidism. Muscles and joints often ache. With respiration impaired and oxygen in short supply, exercise takes a heavy toll on the body, and muscles do not strengthen in response to exercise; nor does stamina improve.

Low thyroid levels actually trigger muscle fibers to change their type, from fast-twitch fibers to slow-twitch fibers. This may be an adaptive strategy for coping with starvation, since blood sugar is low under hypothyroid conditions and fast-twitch muscle fibers require high levels of glucose to operate. Fatty acid levels in the blood are elevated to provide fuel for the fat-burning slow-twitch muscles. However, low oxygen in the blood due to slow heart rate and respiratory problems limits the slow-twitch muscles' effectiveness.

Even after receiving treatment for hypothyroidism, many people find that their caloric needs and ability to handle exercise have changed permanently. Strength training can help restore their fitness, but only after thyroid hormone levels have normalized. Therefore, hypothyroidism affects the ability of people to undergo both aerobic and anaerobic exercise.

Hypothyroidism is the second leading cause of high cholesterol, after diet. When TH levels drop, the liver no longer functions properly and produces excess cholesterol, fatty acids, and triglycerides, which increase the risk of heart disease. High cholesterol may also contribute to the risk of Alzheimer's disease. Hypothyroid patients may develop yellowed skin due to carotenoid (Vitamin A precursors) deposits in the skin when the liver no longer can store enough. Vitamin A usage and synthesis drops as thyroid hormone levels drop.

Effects of TH Imbalance: Hyperthyroidism

Hyperthyroidism is associated with a different set of symptoms. People with this disorder sleep with difficulty and sleep much less than normal. Unlike hypothyroid patients, they exhibit manic-depressive behavior as the TH levels drive their energy levels beyond their physical limits. In fact, thyroid hormone testing is routine at psychiatric admission for suspected manic-depressive patients. Lithium, a common treatment for manic-depression, is known to depress T3 in the brain back to normal levels.

Hyperthyroidism causes accelerated heart rate and fatigue, even when patients are at rest. It

produces lower exercise tolerance because protein and fat catabolism are accelerated, resulting in build-up of ketones. Hyperthyroid people often show a fine tremor in their hands. They have higher resting heart rates, but not higher maximum heart rates for exercise, in comparison to normal subjects. Some experience thyroid storms--high overloads of thyroid hormones that accelerate their heart rate to as high as 300 beats a minute. This is a very life-endangering condition and can result in arrhythmia or heart attack.

Some drugs cause a temporary TH imbalance. Caffeine and other stimulants interfere with T3 and adrenal hormone metabolism while in the body. Smoking depresses TH levels and produces an chronic underlying hypothyroidism as well as low adrenal hormone levels. The hormonal imbalances due to smoking may contribute to the severity of withdrawal symptoms in smokers trying to quit. Research shows that nicotine increases the synthesis of T3 from T4 in the brain, while alcohol and opiates block the breakdown of T3 in the brain[2]. Research into thyroid hormone's role in addiction might lead to better treatment and prevention of drug addiction[3].

Causes of Thyroid Disease

The most common causes of acquired thyroid disorders are iodine deficiency and autoimmune thyroid disease. Iodine deficiency is the major cause of hypothyroidism for much of the world, due to absence of iodine in the diet and/or high consumption of soy, corn, and brassica plants (cabbage, broccoli, brussel sprouts, etc.). These plants produce natural goitrogens. Goitrogens can be largely abolished through proper cooking. In the U.S., salt is iodized to ensure people get enough iodine. Iodine overdose rarely is a problem, as the thyroid gland stores iodine until it is necessary, and releases TH in the less active T4 form, and TH is also bound up by transport proteins in the blood until it is needed. Some experts believe that continual iodine overdoses leads to autoimmune thyroid disease, because it seems to be the major cause of thyroid disorder in developed countries.

Two autoimmune thyroid diseases, Graves' disease and Hashimoto's thyroiditis, are thought to be inherited, but have not been linked positively to any genes. Autoimmune thyroid disease is identified by detecting antibodies in the blood. In the case of Graves' disease, antibodies latch onto an enzyme essential for making T4, and keep it active and continually turned on. Graves' disease is treated by suppressing or killing (removing) the thyroid and then stabilizing the patient on thyroid hormone replacements. In Hashimoto's thyroiditis, antibodies latch onto the same enzyme, but block its function, and help trigger destruction of the thyroid. In the early stages of Hashimoto's thyroiditis, the thyroid may produce too much TH, but as the thyroid is slowly destroyed, the patients TH levels drop. Hashimoto's thyroiditis is treated with thyroid hormone replacements.

Some experts have suggested that autoimmune thyroid disease develops as a result of iodine overconsumption. Both the U.S. and Japan have high levels of iodine consumption and of autoimmune thyroid disease. Japanese people consume iodine because seafood makes up a large proportion of the diet, and Americans do because salt is iodinated and the food industry uses iodine as a machine wash. Other experts believe that pollutants are a more important factor. Pollutant chemicals like polychlorinated biphenyls (PCBs) and dioxins have been shown to interfere with thyroid function and are more prevalent in industrialized countries where thyroid disease levels are high. Autoimmune thyroid disease, either hyperthyroidism or hypothyroidism, is also linked to post-traumatic stress disorder and is often first observed clinically after periods of prolonged stress.

Conclusion

Research on the treatment of thyroid disease is proceeding in promising directions. Autoimmune thyroid disease is being intensively studied, and synthetic antibodies have been produced that neutralize Graves' antibodies in mice. Other studies are uncovering the role of TH in the brain, and finding new genetic causes of thyroid hormone metabolism disorders. TH function is being studied in various vertebrates, and environmental chemicals are undergoing examination as possible TH disruptors. Such research provides hope that autoimmune thyroid disease can one day be attacked at its source.

However, adequate information has not spread into the medical field. Labs performing blood work use overly broad normal ranges of TSH levels. Published research indicates 1-3 ug/ml in the blood (micrograms per milliliter of blood) is the best range of normal[4], but most doctors work under the assumption that values as high as 5.5 are normal, which results in underdiagnosis and undertreatment of many cases of hypothyroidism.

A worse problem is the lack of testing. Though an estimated 200 million people worldwide have thyroid disorders[5], thyroid function tests are rarely given unless the doctor suspects a thyroid disorder, and most doctors do not suspect hypothyroidism in their patients because the symptoms are subtle. Of the estimated 13 million Americans affected by thyroid disease, more than half are unaware of their condition[6]. Thyroid disease affects 8 times as many women as men, possibly because women need higher levels of TH than men do, but it has no age, gender, or ethnic barriers. Patients may have some or all the obvious symptoms: fatigue, lack of focus, depression, constipation, anxiety attacks, dry hair, dry skin, edema (swelling), lack of exercise tolerance, weight gain (especially in the stomach), muscle and joint pains, problems swallowing (due to enlarged thyroid), goiter, facial puffiness, unusual new headaches, loss of eyebrows, lack of sex drive, lowered body temperature, low or high blood pressure, and slowed heart rate. Yet patients may not be diagnosed for years.

The link between high cholesterol and underlying hypothyroidism is vastly overlooked, even though cholesterol's role in heart disease is heavily publicized. People have their cholesterol tested more regularly than their thyroid hormone levels. The result is prescriptions for expensive cholesterol-lowering drugs that don't address the real problem. People diagnosed with high cholesterol, especially those with low body temperature, should have their thyroid function tested before they begin taking such drugs. Also, smokers and other substance abusers should be watched for hypothyroidism (and urged to quit), as stimulants and depressants both can affect TH metabolism.

The under-diagnosis of thyroid disease handicaps research as well as the lives of affected patients. Researchers need to understand the proper function of thyroid hormone and the pathology of thyroid disease to fully understand how our bodies, brains, and immune systems develop and work, in health and in illness. It is impossible to know the prevalence of thyroid disease and figure out all the causes if patients take years on average to be diagnosed. We still do not know what causes the high prevalence of autoimmune thyroid disease in developed countries. Until researchers turn up strong and clear evidence on the cause, more cases of autoimmune thyroid disease will occur every year.

1. Synthroid, Knoll Pharmaceutical Company (http://www.synthroid.com/consumer/1310.htm)
2. Examination of antithyroid effects of smoking products in cultured thyroid follicles: only thiocyanate is a potent antithyroid agent (Acta Endocrinol (Copenh), 1992 Dec, 127(6):520-5)
3. Thyrotropin releasing hormone decreases alcohol intake and preference in rats (Alcohol, 2000 Jan, 20(1):87-91)
4. Weetman AP. Fortnightly review: Hypothyroidism: screening and subclinical disease. BMJ 1997;314:1175 (19 April) (http://www.bmj.com/cgi/content/full/314/7088/1175)
5. Thyroid Foundation of Canada (http://www.thyroid.ca/Guides/tg00.html)

back to article

6. American Association of Clinical Endocrinologists (http://www.aace.com/pub/aacc/tam2001/acesstam2001.html)

EXHIBIT C

alt.support.thyroid
T3 Supplementation

The T3 Story

T what?

Let's hear you pronounce it: triiodothyronine. There, now you know why the name is usually shortened to T3. T4 (thyroxine) and T3 are the main thyroid hormones. T3 is five to eight times as strong as T4 (taking into consideration that it's absorbed at a higher rate than T4), and it's biologically more active. T4 is like the food in your refrigerator, while T3 is like the food on your plate.

T4 is slow acting, with a half-life of about one week — after a week, you have about half the level of the T4 still in your body, a week or so later you have half of that half remaining, and so on. Its full effects aren't reached until about six weeks after starting or changing a dose, which is why lab tests are optimally done every six weeks or so until a patient with hypothyroidism has reached satisfactory and stable thyroid hormone levels. T3, on the other hand, has a half-life of about a day. People on T3 sometimes feel its effects within minutes after taking it.

T3 is available as a separate synthetic medication with the brand name Cytomel in the US and Canada, and Tertroxin in the UK. It's usually prescribed along with a synthetic T4 medication. In the US, a synthetic T4/T3 combination is available with the brand name Thyrolar.

Natural, **desiccated thyroid** from pigs' thyroids with the brand name Armour is sold in the US, and in Canada, desiccated thyroid with the brand name Thyroid is made by Erfa (formerly by Pfizer and before that, by Parke-Davis). Westhroid and Nature-throid are available in the US, and Natura-throid is also available in Europe.

T3 or not T3

A thyroid gland that functions normally produces T4 and T3. Twenty percent of the T3 circulating in the body comes directly from the thyroid gland, and the remaining 80 percent comes from conversion of T4. Because of this conversion process,

T3 Files

T3 Supplementation
An explanation of the thyroid hormone T3 (triiodothyronine) and why supplementation of T3 along with T4 (thyroxine) is extremely beneficial to many people with hypothyroidism

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T3 References
An overview of the references in this section

From medical journals and associations

1. T4/T3 Combination Therapy and Euthyroidism
Web page
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2. T4-to-T3 Conversion and Hypothyroidism
Web page
Printer-friendly

3. Hypothyroidism, T3, Mental Function, and Depression
Web page
Printer-friendly

4. Hypothyroidism, T3,

most doctors prescribe only synthetic T4 medication (Synthroid, Levoxyl, Levothroid, Eltroxin, Unithiold, and others). Many patients with hypothyroidism do fine on T4 only.

However, many others don't, and they need T3 supplementation in addition to T4. If the thyroid gland is malfunctioning and not producing enough — or any — T4, why assume that it still puts out enough T3, or that the body converts enough of its T4 to T3?

The addition of T3 often helps with many symptoms of hypothyroidism that may not disappear with supplemental T4 only. It has improved people's libido, memories, and vision. It has eliminated or greatly reduced brain fog, feeling cold, constipation, depression, chronic fatigue, headaches, insomnia, muscle and joint pain, and chronic sinus infections. For some people, but not all, it has helped them finally lose weight. A small percentage of people who try it feel worse or no better on it.

T3 tests

Do **lab tests** show if a person needs T3 supplementation? Sometimes. If free T3 is lower in its range than free T4 is, this suggests that more T3 would be beneficial for that person. On the other hand, some people who have posted in alt.support.thyroid have had lab results that did not indicate a problem with T3, but they still had symptoms of hypothyroidism, and the addition of T3 helped them.

Lab results do not tell the whole story. However, most people with hypothyroidism in alt.support.thyroid feel best when their free T4 and free T3 levels are in the upper part of their ranges. The exception is with people who are on desiccated thyroid. Because it contains a higher ratio of T3 to T4 than our thyroids produce, people taking it have a free T4 level that's lower in its range when the free T3 level is where it should be, in the upper part of its range.

Tell my doctor

Doctors are taught in medical school that T4 is the only thyroid medication that patients with hypothyroidism need. For many patients, that's true. The problem is that many other patients are left with reduced quality of life on T4 only — but their health improves greatly once T3 is added.

The medical establishment is increasingly looking at T3 in

and Heart Disease
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From other sources

1. From doctor-written articles
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2. From interviews with doctors
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3. From websites other than the above
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4. From books
Web page
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Patients' Experiences

Printable compilations of patients' own words about how they felt after they started taking T3
Improved Life
Mental State
Symptoms

addition to T4 as essential treatment for some hypothyroid patients. Not having been educated on its use, however, some doctors are hesitant to prescribe it. That's why we've compiled **references** from medical journals and other sources that discuss the effectiveness, safety, and necessity of T3 supplementation for many patients with hypothyroidism.

To find a doctor who prescribes T3 for some patients, see our **tips on finding a good thyroid doctor**. To try to convince your current doctor to prescribe T3, you can bring medical references (see the column at the right) or send them to your doctor before your appointment and ask your doctor to prescribe at least a trial amount of T3.

Tell me more

See the links at the right on this and all the pages in the T3 section of this site. The **T3 References** page provides an overview of the references and why we need them. The compilations of references are divided into mecical journal references and references from other sources, and each has a printer-friendly version so that you can take these references to your doctor if they're relevant to your situation.

The above list of symptoms that T3 has helped with is from the "T3: Patients' Experiences" compilations.

If you want to discuss T3 supplementation with your doctor, we recommend that you read through the files here, and perhaps print copies of them for your doctor. Because T3 is so much stronger and faster acting than T4, it's important to get the doses right. On pages 285 and 286 of his book *The Thyroid Solution*, Dr. Ridha Arem describes how to adjust T4 doses when supplementing T4 with T3. Some people in our group have brought this book to their doctors, and their doctors have used this protocol to successfully add T3 to the medication mix.

Kevin G. Rhoads discusses TSH, T3, and T4 in more detail in his **Thyroid 101 and Basic Fallacies** post. See also the related articles **The Desiccated Thyroid Story** and **The TSH Story** as well as **Hypothyroidism Medication** and **Hypothyroidism Medication Comparison**.

Lois Summers

This page was last updated June 14, 2008.

PATENT

IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

Application No.:	10/535,215
Confirmation No.:	2007
Filing Date:	May 17, 2005
Applicant:	Jan SVENSSON et al.
Title:	FUNCTIONAL CEILING SYSTEM
Attorney Docket:	315-0039/US

Customer Service Window
Randolph Building
401 Dulany Street
Alexandria, VA 22314
Mail Stop PETITION

September 11, 2009

STATEMENT BY MR. HERMAN R. HEFLIN III

Sir:

In support of the Petition to Revive Under 37 CFR § 1.137(b) submitted concurrently herewith, please consider the following information.

1. On September 4, 2009, Applicants' Swedish representative, Ström & Gulliksson AB ("S&G") instructed me, Herman R. Heflin III, to take over responsibility for United States Application No. 10/535,215 ("the '215 application").

2. Beginning September 4, 2009, I began to review the prosecution history of the '215 application using the USPTO's PAIR system.

3. S&G and I discussed the circumstances surrounding the delay in responding to the January 12, 2006 Notification of Missing Requirements. Based on those discussions, I prepared the Petition to Revive Under 37 CFR § 1.137(b), and this Statement.

4. In connection with preparing the Petition, I contacted Mr. Steven S. Payne directly regarding his Statement, which is submitted concurrently herewith.

5. I declare that all statements made herein of my own knowledge are true, and that all statements made on information and belief are believed to be true. These statements were made with knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under Section 1001 of Title 18 of the United States Code, and that such

willful false statements may jeopardize the validity of the application or any patent issuing thereon.

Respectfully submitted,

/Herman R. Heflin III/
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